Cholesterin is very poisonous when it is in quantity slightly above the normal. The bile is probably the means by which it is eliminated. The presence of aromatic substances in the blood is not poisonous. The asphyxial form of acute alcoholic poisoning is probably due to the transformation of alcohol into aldehyde. *Lancet*, February 2, 1884.

MEDICINE.

Limited Laryngeal Paralysis.

IN a recent number of the *Berliner klin. Wochensch.*, has appeared a long paper by Dr. FELIX SEMON, in which he gives an able review of the facts bearing upon the assertion, which he was the first to make some three years ago, that in all cases of organic disease or injury of the motor nerves of the larynx there is either palsy of the abductor muscles alone, or these muscles are affected earlier and more severely than any others. At the time of his first paper, published in the *Archives of Laryngology*, July, 1881, Dr. Semon was able to report twenty-two cases, completed by autopsies in ten instances, supporting this statement; he now adds thirty-six new cases with eleven autopsies, making a total of fifty-eight cases, of which, in twenty-two, a careful autopsy has been made. These cases have also been observed by such competent authorities as Gerhardt, Riegel, Penzoldt, Mackenzie, Moxon, Hilton Fagge, Ord, Dreschfeld, Remak, F. Taylor, Whipham, and others. They include cases of encephalitis, cerebral syphilis, disseminated cerebro-spinal sclerosis, general paralysis, apoplexy, disease of pons and medulla, tabes dorsalis, lead-poisoning, goitre, cancer of the thyroid, enlargement of the bronchial and cervical glands, pulmonary tuberculosis, chronic pneumonia, aneurism of the aorta and of the innominate artery, lympho-sarcoma of the anterior mediastinum, cancer of the œsophagus, and typhoid fever. Various as are these primary diseases, it is shown that in all of them, when either of the motor nerves of the larynx—the spinal accessory, pneumogastric trunk, or recurrent branch of it—is affected, the palsy involves primarily, chiefly or solely, the abductor muscles of the glottis. Schnitzler and Solis Cohen, Jr., have recorded cases in which they allege there was isolated adductor paralysis in organic disease of the motor laryngeal nerves. But after a careful review of these reports, Dr. Semon shows, in part from personal observation, that they are based upon insufficient evidence, and must not be held to invalidate at all his conclusions based upon a much larger number of cases and corroborated by many autopsies. In not one of the four cases of Schnitzler or Solis Cohen was there an autopsy made. It is generally admitted that in cases of soft lymphatic goitre isolated and varying paralysis of the adductor muscles may occur. Schnitzler attributes this to pressure of the enlarged thyroid upon the recurrent laryngeal nerves, but Semon holds that this palsy is functional, like all other isolated adductor palsies, and is to be attributed to transitory anæmia of certain regions of the brain; possibly this view gains support from the remarkable results of excision of the thyroid gland recorded by Kocher.

It is impossible to deny the force of the facts that Dr. Semon urges in support of the generalization which he himself fairly claims to have originated. The cases he records are alone sufficient to show the great practical importance of it. For it must be remembered, and this is by no means generally appreciated, that pure unilateral paralysis of the abductor muscle of the glottis does not interfere with respiration or with the voice, and its existence may be, and often is, overlooked, simply because it is held to be unnecessary to examine the larynx thoroughly in the absence of special symptoms pointing to its affection. But Semon has demonstrated that the recognition of this isolated abductor palsy, which is often a very early symptom of obscure and grave organic disease, may be taken as absolute proof of the existence of such organic affection, and thus enable the practitioner to arrive at a diagnosis earlier than would be otherwise possible, and save him from errors in prognosis. Indeed, the facts warrant the examination of the larynx in all cases of disease of the brain, neck, and chest, in which the laryngeal nerves can be affected either at their centres or in any part of their course. To physiologists, however, Semon's law is of the deepest interest and importance; for it points to the conclusion that the ganglionic cells composing the nucleus of the spinal accessory nerves are strictly differentiated. Buzzard and Ormerod have shown reason for the belief that the same holds true in relation to the oculo-motor nerve centre; and it therefore becomes probable that the cells of the nuclei of all cranial and spinal nerves are strictly and exactly differentiated. The special proclivity of the abductor nerve-fibres to succumb to general disease, too, must not be regarded as a pathological curiosity, met with only in the larynx, but as the expression of a general law, which has been formulated by Ferrier, that all abductor and extensor nerves possess less vital resistance, and are sooner exhausted than adductor and flexor nerves. Dr. Semon does not in his paper enter upon the physiological explanation of the phenomenon as regards the larynx, but has reserved this for another occasion. The subject is one that by no means specially concerns so-called laryngologists, but, as the list of cases we have quoted shows, interests far more general surgeons and physicians, and moreover illustrates the importance of medical practitioners being able to investigate thoroughly all the organs of the body.—*Lancet*, Dec. 8, 1883.

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Sciatic Neuritis.

An account is given in the *Gaz. des Hôpitaux* (No. 16), of a case of this description which came under Dr. Landouzy's care at the Charité. A woman, 33 years of age, but apparently much older, applied on account of an affection of the chest, and on examination manifested all the signs of extensive tubercular infiltration of the left lung. There was also remarkable atrophy of the right leg, and although the woman did not apply with respect to this, she admitted that for the last 18 months she had constantly suffered severe pain in this limb, whether she was using it or not, and at night as well as by day. The seat of pain was referred to the sciatic nerve at its exit, and along the course of its trunk and principal branches. This peculiar course and continuity of the pain were pointed out by the late Professor Lasègue as the means of diagnosing between sciatic neuritis and neuralgia, and Dr. Landouzy did not hesitate to attribute the present case to the former affection, especially as it was also accompanied by marked atrophy of the limb. As to the relation of the neuritis to the tubercular infection, it is to be observed that the patient, who had no family or personal history of phthisis, had cohabited with two men in succession who were the subjects of this disease; but she did not complain of her health until 18 months before her admission, when she became the subject of peritonitis, and afterwards of fistula in ano, for which she underwent operation. It was during this period also that the painful affection of the leg commenced, and that she was seized with cough and hæmoptysis. Dr. Landouzy does not hesitate to regard the neuritis as one of the localizations of tubercle, of which other examples are on record. Professor Peter, for example, in his *Clinique Médicale*, has shown by several examples that disturbances in innervation of the highest importance may be produced in phthisis, and that they are associated with its most serious forms, or appear at the moment of definitive and hopeless aggravation of the tubercular disease—becoming an indication of the extreme gravity of the disease and of the approaching death of the patient. He refers to two such cases of sciatic neuralgia, or rather neuritis; at his instigation, M. Friot produced, in 1879, a thesis in which this point in the history of tuberculosis was examined. Among 137 cases of phthisis admitted into Professor Peter's wards in 1878, M. Friot met with four instances of the occurrence of sciatica in the course of phthisis, which, joined to Professor Peter's two cases, and five others reported elsewhere, form the material for his conclusions. In Professor Peter's two cases, and in three of those related by M. Friot, as well as in the one which forms the subject of his paper, the sciatica closely coincided with the commencement of the pulmonary tuberculosis. His conclusion is that sciatic neuritis depends upon a special anatomical lesion, such as tuberculosis, influencing the nervous substance or its coverings and acting upon neighbouring nerves. Two of his cases tend to prove that in tubercular sciatica there is always a changed condition of the spinal cord or its membranes, a condition of a variable character, but capable of producing secondarily a peripheral neuritis. Summing up the results of M. Friot's eleven observations, it seems that this neuritis may commence the series of affections asssociated with phthisis, or burst out during the course of the disease. The pains in place of being intermittent as in neuralgia are constant and very obstinate; and their appearance should give rise to the suspicion of tubercular change either in the cord or its membranes, or in the spinal column. They tend to aggravate the disease, and generally precede only by a short time the fatal termination. While in sciatic neuralgia we may obtain a cure by means of narcotics and antispasmodics, antiphlogistics furnish the only means of relieving the pains of neuritis, while a complete cure is in all cases unattainable.—*Med. Times*, Jan. 24, 1884.

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Angina Pectoris Immediately Followed by Pericarditis.

At the meeting of the Clinical Society of London, on January 25, 1884, Dr. DONALD HOOD related the case of a gentleman, æt. 65, who, while resting on a sofa, apparently asleep, was suddenly aroused by the window curtains being on fire. He quickly jumped up to extinguish the flames, and within half an hour after this exertion he was suddenly seized with severe heart-cramp. Seen shortly afterwards by Dr. Hood, he was noticed as being blanched, barely able to speak, heart irregular and fluttering, pulse of peculiarly low tension, with agonizing cramp-like pain over cardiac region. Hot stimulating applications, brandy, ether, and opium, were used freely, and the patient slowly lost the pain, and a general improvement of the circulation took place. Twenty-four hours after the commencement of the attack a soft pericardial shuffle was noticed; within a few hours it had deepened into a distinct rub. From this time during the succeeding ten days the case assumed all the characters of one of pericardial effusion; the amount of fluid being but moderate, and apparently completely absorbed within the ten days. A fortnight later the patient was found to have a systolic apex murmur, and symptoms of incompetency of the mitral valve rapidly developed. The patient died four months later from syncope, and at the time of death was the subject of cardiac dropsy. No *post-mortem* could be obtained. Dr. Hood suggested, as a possible explanation of the primary attack, with its subsequent train of symptoms, that the patient was the subject of some fibroid degeneration of the heart. Roused from sleep by the flames, his alarm and exertions caused a sudden distension of the heart cavities, which possibly gave rise to some lesion of the visceral layer of pericardium, and so started the pericarditis. In support of this hypothesis, Dr. Hood called attention to the series of cases of fibroid disease of the heart, collected and published by the late Dr. Fagge in the twenty-fifth volume of the Transactions of the Pathological Society, and stated that in more than half of these cases the patients were found to have been the subjects of pericarditis. He further suggested that fibroid degeneration of the heart should be looked upon as a possible factor in those cases of pericardial effusion in which it was difficult to explain the cause of the pericarditis.

Dr. DE HAVILLAND HALL gave the details of a case seen by him five years ago, in which a man, aged 56, had suffered from severe pain in the præcordial region shortly after a violent and passionate altercation with his son. The pain was still present on the following day, but was somewhat relieved by the use of diffusible stimulants, and of morphia subcutaneously, but never quite disappeared. The sounds of the heart were then normal, and there was no rise of temperature. Within forty-eight hours signs of pericarditis became manifest, a moderate effusion took place, the patient became worse, and died in four or five days.

Dr. DUCKWORTH said that hitherto no clinical connection had been recognized between cases of genuine angina pectoris and pericarditis by any of the standard authors. The classical disease, angina pectoris, was generally relieved by the well-known remedies. But there were other forms of angina, and especially in connection with aortic disease, in which severe cardiac pain was set up, and he considered it possible that the cases related might have been of this class. Chronic mediastinal inflammation, disturbing the nerves of the part, might give rise to cardiac neuralgia. These cases, however, were different from those of true angina pectoris. He thought that probably very few physicians had seen many cases of the latter affection, whilst the occurrence of cardialgia must be familiar to most observers. Simple cardiac neuralgia, however, was not usually followed by pericarditis.

Dr. W. EWART thought that it would be of interest if evidence could be brought forward of the extent to which pathological changes on the heart and great vessels could give rise to symptoms. He had met with many instances of cardiac pain simulating neuralgia, brought on simply by excitement. It would be difficult to explain the occurrence of pericarditis from a purely functional cause; but he thought it possible that there might be an enlargement of the aorta within the pericardium, which, escaping detection, might suffer a slight rupture and so set up pericarditis. He would be inclined to attribute the sequence of events in these cases to some such coincidence.—*Med. Times and Gazette*, Feb. 2, 1884.

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Interstitial Chronic Idiopathic Hypertrophic Myocarditis.

PETRONE reports the following case: A man, æt. 25, admitted after six months' illness, had not suffered from rheumatism, presented œdema of the ankles, moderate dyspnœa, cough and mucous expectoration; small, weak, quick pulse; sounds and impulse of heart feeble; liver enlarged. After a month's treatment with digitalis, quinine, etc., all these symptoms improved; but they soon returned in an aggravated form, with progressive enlargement of the cardiac dulness, pulmonary congestion and œdema, and a systole. *Post-mortem*, there was found to be hypertrophy of the heart, especially of the left side; the orifices and valves were healthy; the peripheral vessels were not atheromatous; the aorta was slightly atheromatous. On section, the cardiac muscle was normal in colour and hardness; examined histologically, the connective tissue separating the fasciculi of muscle-fibres was considerably increased. These fibres were so resistant, that with the forceps it was possible to detach the muscular elements and to isolate the connective stroma. This tissue appeared so developed as to form extensive fibrous plates of various configuration, in the midst of which were seen the muscular fibres in various stages of atrophy. In the centre of each patch was found a small artery, surrounded by connective tissue, and radiating towards the periphery; the artery presented all the characteristics of endoperiarteritis obliterans. This history shows, according to Petrone (*Lo Sperimentale*, and *Annali Univ. di Med.*, Nov. 1883), that there exists a primary isolated cardiac cirrhosis, without renal lesion; the cause of this myocarditis being a diathesis, probably determined by cold and damp or alcoholism. The author quotes the opinion of Rigal, according to whom the alterations produced by the diathesis or intoxication (tobacco, gout, rheumatism, alcoholism, or lead-poisoning) are manifested primarily in the small arteries and capillaries of the cardiac muscle. The suppressed elasticity of these vessels creates an obstacle to the movement of the heart, and hence the hypertrophy. This hypothesis is probable, but not certain. The diathesis may display its influence both on the interstitial connective tissue of the heart-muscle and on the arterial walls, the hypertrophy being the effect of the diffused sclerosis of the myocardium. Certain symptoms render the diagnosis of this form of sclerosis possible: hypertrophy, weakening of the systole, absence of murmurs, disproportion between the enlargement of the area and the weakness of the cardiac impulse and radial pulse, the impossibility of deciding for any other disease of the heart, the absence of polyuria, attacks of pulmonary congestion which (as Rigal admits) depend on vaso-motor paralytic disturbances, slight dropsies, which disappear to reappear quickly, and the absence of albuminuria. Joined to a systole, it is impossible to distinguish it from other heart-disease. Treatment is impotent against this form, which inevitably leads the heart-muscle to progressive degenerative atrophy.—*London Med. Record*, Feb. 1884.

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Hemorrhage from the Hepatic Vein—Sudden Death.

DR. ALFRED F. HOLT reports the following interesting case:—

J. C., æt. 56, died May 15, 1883. For the last six or seven years of his life he had been engaged in a small kindling-wood trade, doing some of the manual labour required by such a business himself. For the ten years before this time he had been a police officer.

Ten or twelve years before his death he began to have severe attacks of colic. These continued to occur at irregular intervals during his life, and for the last six or seven years he was never quite well, although able most of the time to attend to his business. During these years he had consulted a large number of physicians. Most of these pronounced his trouble gall-stones, but some of them dyspepsia, and still others some obscure disease of the liver.

Several days before his death he was suddenly prostrated with a very severe attack of pain in the region of the stomach, nausea, and faintness. He was taken to his home and his physician, Dr. Dow, who was immediately summoned, found him suffering from severe pain, vomiting at short intervals, pale, and faint. The pain and faintness continued until evening, he having been taken sick about noon, when the doctor was hastily called again. He found him vomiting blood in the form of very large dark clots. This continued at short intervals for about one hour, when it ceased. A little over three pints of blood were vomited at this time. The pain and faintness continued for three days. Although these symptoms were somewhat relieved by stimulants and opiates, at the end of this time he was again attacked with the vomiting of blood, as before, in the form of very large black clots. This continued at short intervals until nearly two quarts had been ejected, when it suddenly ceased; and after this he was very weak, faint, and nearly pulseless. The pain continued a few hours after this vomiting, when it ceased. Although his mind was clear, he did not rally, but gradually sank, and died forty-eight hours later, or a little less than seven days from the commencement of the sickness. No blood was at any time passed by the rectum.

Autopsy.—The heart was seen to present a noticeably peculiar appearance, the right ventricle being distended so as to make a rounded prominence on its upper part near the auriculo-ventricular junction. The right auricle was also distended. Both of these distensions felt elastic to the touch. On opening this side of the heart there was an escape of a puff of gas, and the walls of the organ immediately collapsed. There was no blood in either of the cavities. The right ventricle was a little dilated, and its walls were slightly thinner than normal. Left side of heart empty. The whole organ was rather large. Its tissues were pale, but of a uniform colour. The valves were in every way healthy, and but little blood flowed from the cut ends of the large vessels.

The common bile-duct increased in calibre as it approached the liver, until it and the hepatic duct were dilated into a sac or pouch of about two ounces capacity. The walls of this pouch were very thick and hard, and there was a deposit of a dense tissue around them. At the lower side of this sac, near where the gall-bladder entered it, was a cup-shaped depression the size of half a chestnut. When a little water was poured into this a shreddy mass floated up, being attached to the sides and bottom (recent ulceration). On the upper side of this dilated duct, and immediately opposite where the bloodvessels entered the left lobe of the liver, was a similar spot of ulceration, somewhat larger and irregular in outline. On handling, a little blood was seen to flow from the bottom of this depression, and an opening was readily found leading directly into one of the vessels entering the left lobe of the liver.

This was believed to be a branch of the hepatic vein, but all of the tissues in this vicinity were bound together in such a confused mass that it was not quite certain. The gall-bladder was much thickened and contracted in such a way as to make its cavity very irregular, and of a capacity of not more than two or three drachms. Its mucous surface had wholly lost its honeycombed appearance. The opening between it and the sac was wide enough to readily admit the finger. Both the gall-bladder and sac when opened contained a little bloody fluid.

The cut surface of the left lobe of the liver was of a uniform greenish-brown slate colour, smooth but not glistening; and the contrast between it and the right lobe was very striking. It felt firm. The cut surface was of the same uniform greenish-brown colour. All appearance of structure had disappeared. A piece of this lobe could readily be broken with the hands; and the fractured surfaces had much the appearance of nearly dried piece of mud treated in the same way. The larger bloodvessels could be made out in the mass. They were filled with a dryish brown-red material that remained formed when turned out of the vessel, but readily crumbled in the hand into a coarsely granular mass. The main vessels entering this lobe of the liver near the ulceration above described were filled with the same material.

Unquestionably the source of the hemorrhage was the open bloodvessel supplying the left lobe of the liver; the blood readily finding its way into the duodenum by means of the largely dilated duct and its opening into the intestine, and through the rather open pylorus into the stomach. Why all of the blood should take this course and none of it go down the intestinal canal, as it apparently did not, is one of the points in this case seemingly worthy of discussion.

Of course the gas accumulating in the intestinal canal after death found its way by means of the same open vessel to the right side of the heart.

Hæmatemesis, with the blood coming from such a source, must be exceedingly rare, and so far as Dr. Holt can learn this case is unique.

The pecular condition of the left lobe of the liver he has never seen before. He believes it to have been caused by a complete cutting off of the blood supply, producing a necrosis of the part.—*Boston Med. and Surg. Journ.*, Dec. 20, 1883.

Icterus Neonatorum.

A recent number of the *Zeitschr. f. Geburtsh. u. Gynäk.* contains an article on the above subject by Dr. M. HOFMEIER, of Berlin. The writer first considers the theories hitherto advanced as to the production of this condition. It has been suggested, first, that it is due to changes taking place in the blood, leading to production and non-elimination of pigment. Dr. Hofmeier does not accept this view, because he finds no evidence of the supposed blood changes, or of the assumed deficiency in the emunctory organs. It has been also said to be due to pigmentary changes in capillary cutaneous extravasations produced during labour. Three points, says our author, tell against this—(1) there is in this disease coloration of internal organs as well as of the skin; (2) it is met with in small and premature children, as well as in those with whom labour has been protracted; (3) it is met with in children delivered by Cæsarean section, or by laparotomy, in cases of extra-uterine pregnancy. A third theory is, that it is due to diminution of blood-pressure generally, and therefore in the portal system, leading to re-absorption of bile. To this Dr. Hofmeier objects (1) that there is no proof that lowering of the blood-pressure does lead to re-absorption of bile; (2) that if it were so, in infants in whom the late ligature of the cord is practised, so that the blood-pressure in their circulation is little diminished, or even increased, jaundice ought not to be met with; whereas it occurs as commonly in them as in others. Virchow supposes, and so does Kehrer, that icterus neonatorum is due simply to catarrhal swelling and occlusion of the bile-duct. Dr. Hofmeier accepts this as true for a few cases, but not for all. The latest explanation is that of Birch-Hirschfeld, who, basing his views on 600 post-mortem examinations, attributes the jaundice to compression of the bile-ducts in consequence of venous stasis in the liver. Dr. Hofmeier points out that such venous stasis implies grave disturbance in circulation and respiration; and while entirely accepting Birch-Hirschfeld's facts, he yet sees jaundice so often in children whose breathing and circulation are quite vigorous, that he thinks the post-mortem appearances in children who die do not account for the jaundice in robust children who live.

Finding, thus, that none of the explanations of icterus neonatorum hitherto advanced explain its occurrence in a satisfactory manner, Dr. Hofmeier has himself investigated the subject from a clinical point of view. He finds that with the jaundice of the new-born the following phenomena are associated: During the first few days of life the loss of weight which occurs in all children is in the icteric much greater than in others. There is also during the first nine days a greatly increased excretion of urea and of uric acid; and there are also appearances in the urine which (from a former investigation) he believes to indicate the presence of uric acid infarcts in the kidneys. Albuminuria occurs more often in the icteric than in the non-icteric. In the jaundiced a yellow pigment is invariably present in the urine, to a degree corresponding to the intensity of the jaundice. These things indicate, he thinks, a considerable waste of the nitrogenous tissues; and this inference leads to the next question, viz., as to the cause of the waste. It might be due (1) to deficient quantity of nutrition, (2) to defective quality, (3) to mal-assimilation. These disadvantageous circumstances would be expected to more affect first-born and premature children, and those whose mothers were suffering from illness. Observation corroborates theory, for Dr. Hofmeier finds from statistics that among icteric children there are undue proportions of first children and of premature children, and that icterus is more often seen in lying-in hospitals than in private practice. Assuming, then, that there is an excessive waste of nitrogenous constituents, the question comes—on what tissue does this fall? Dr. Hofmeier answers—on the blood. It leads, in his view, to destruction of red blood-corpuscles; and it is the pigment derived from this disintegration that causes the jaundice. He points out that many blood-poisons—both chemical, such as phosphorus, and vital, such as the poison of pyæmia—produce jaundice as one of their symptoms; and some among these (*e. g.*, ether and chloroform) have been experimentally proved to cause destruction of red blood-corpuscles. Dr. Hofmeier has sought for direct proof of his theory by examining the blood. In the newly-born he finds these peculiarities present: less tendency to the formation of rouleaux, a greater resistance to the action of fluids which make the corpuscles swell, and a great variability in the number of white corpuscles. These changes he finds more marked in the icteric than in the non-icteric children.

From these facts Dr. Hofmeier draws the general conclusion that icterus neonatorum is intimately connected with certain phenomena depending upon tissue-changes, these changes themselves depending upon the state of nutrition; and further, that a connection between icterus and certain processes going on in the blood is unmistakable. The jaundice he believes due to the presence of bile in the blood, bile-pigment being present in the excreta to an extent proportionate to the depth of the jaundice. Bile-pigment he regards as physiologically an ultimate product of the decomposition of blood-pigment. He thinks that there is an excessive production of very highly pigmented bile in consequence of the nutritive changes previously mentioned, and that the jaundice results from the re-absorption of this. Icterus neonatorum, therefore, according to Dr. Hofmeier, depends essentially upon the state of nutrition during the first few days of life. It is a partly physiological, partly pathological phenomenon; and when it is observed to be intense and of long duration, it should be regarded as an indication of some radical fault in nutrition, and the especial attention and care of the medical man should be directed to the discovery and removal of that which is exerting an unfavourable influence.—*Med. Times and Gaz.*, Dec. 8, 1883.

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Rupture of the Vermiform Process.

M. Polaillon, Surgeon of the La Pitié, communicated to the Society of Medicine of Paris (*Union Méd.*, January 3), an interesting case of rupture of the ileo-cœcal appendix during an effort. A washerwoman, 19 years of age, and in the enjoyment of perfect health, while trying to lift a large bundle of linen, was seized with a sudden pain in the abdomen, accompanied by the sensation of something bursting. The pain kept increasing, together with great tenderness in the abdomen, especially in the right iliac fossa. Symptoms of intestinal obstruction appeared, and as no relief was obtained by the measures adopted, the patient was brought to La Pitié on the seventh day after the commencement of the symptoms. By this time her situation had become very desperate, and an incision was made in the left inguinal region, in order to search for the seat of obstruction. A discharge of fetid sero-purulent fluid announced that the intestine was already perforated, and that purulent peritonitis existed. Further exploration was not pursued, and an artificial anus was formed. The patient died ten hours after the operation, and abundant adhesions and suppuration were found at the autopsy. The vermiform appendix, in an inflamed condition, was found adherent at the upper aperture of the pelvis, and near its free extremity was an oval ulceration, in the centre of which was a rounded perforation; and a little distance above the ulceration a raw bean was found obstructing the calibre of the appendix, but movable towards the cœcum.

M. Polaillon observes that the case is remarkable as an instance of the rupture of the appendix by a mere effort, without any precursory signs, the subject being quite well at the time. Still, even in the absence of symptoms, it is evident that the foreign body had effected such a change on the coats of the appendix as to diminish their power of resistance and prepare the way for a rupture. The case also shows that peritonitis may give rise to the three symptoms, gaseous distension of the abdomen, fecal vomiting, and the absence of the emission of gas *per anum*, the union of which symptoms generally indicates intestinal obstruction. The error in diagnosis, however, is more apparent than real, for the adhesions arising from peritonitis may readily cause obstruction of the intestinal canal.—*Med. Times and Gazette*, Feb. 16, 1884.

The Forms of Purpura.

Dr. **Henri Leloir**, in a recent paper on this subject, groups the pathogenic causes of purpura in the following manner:—

A. *Purpura from Modification of the Vessels.*—1. By disturbance of the capillary circulation, whatever may be the origin, causing active or passive hyperæmia, and producing, according to the case, hemorrhage by diapedesis or by rupture of the vessels. Nervous purpura should be included in this group. 2. Telangiectasic purpura, or the purpura of Cornil. While the pathogenesis of this form of purpura is not yet well determined, it nevertheless constitutes a special class by its pathological and clinical characters. In the case of Cornil and Frémont there was no extravasation of red globules, in spite of the existence of all the clinical characters of the petechiæ. 3. Purpura by primary alteration of the vascular walls, and consecutive rupture of these walls.

B. *Purpura by Modifications of the Blood.*—1. By too great fluidity of the blood. If the existence of this group was demonstrated there would be a purpura by diapedesis. 2. By vascular obstructions determined by certain elements contained in the blood, and causing the formation of thrombi or emboli. This purpura would be caused by simple diapedesis. It appears to be caused more often by vascular rupture; it is more often a hemorrhagic infarction of the integument. *a.* By formation of clots in cases of blood dyscrasia. *b.* By emboli formed of white blood globules (leucocythæmia, etc.). *c.* By emboli formed by bacteria or micrococci (septic affections, etc.). *d.* By emboli formed by altered blood globules.

C. *Nervous Purpura.*—It is very probable that cases are sometimes observed which cannot be placed in any of the above-mentioned groups of purpura. Very often the origin of purpura (leaving aside certain forms of nervous or embolic purpura) seems to be complex. In a number of cases a detailed examination of observed phenomena shows that different pathogenic causes coexist, and it should never be forgotten that the pathogenesis of a case of purpura may be complex.—*Annales de Dermat. et de Syphil.*, Jan. 1884.

Forms of Skin Diseases in Diabetics—Diabetic Dermatosis.

Kaposi has recently published a paper on this subject, which was read before the Vienna Medical Society. He has lately observed a peculiar form of inflammatory gangrene of the skin, for which he proposes the name diabetic bulboserpiginous gangrene. The case was that of a woman, æt. 51 years; early in May, 1882, a gangrenous inflammation appeared in several places on the left leg. This gradually became worse until January, 1883, when Kaposi saw her. There was an elongated gangrenous spot, of triangular form, on the left leg. A second gangrenous spot as large as a five-franc piece was situated below and posterior to the spine of the tibia. A third small spot was situated at the middle of the external border of the calf of the leg. On the anterior face of the leg, and somewhat surrounded by the gangrenous spots, were fifteen or twenty bullæ, whose size varied from that of a pea to that of a bean; they were oval or round, somewhat painful, and contained a muddy material. Some of them were broken, and below the skin was covered with a yellow eschar.

The three gangrenous points presented, then, the following common characteristics. The convex border was encircled by a corona of flabby bullæ, the contents of which were muddy-looking. In the first third there was dry softening, they were pulpy in the middle third, and in the third next to the convex border there were exuberant vegetations in process of cicatrization. The skin situated between the gangrenous spots and the bullæ was normal. There was no fever.

It is evident, then, that the gangrene caused certain isolated bullæ, and that its progress was caused by the production of bullæ, which process is opposed to separation, and it is equally evident that the character of the process is that of a bullo-serpiginous gangrene.

In this case the analysis of the urine showed 5.15 per cent. of sugar; there was no polyuria or polydipsia. Under the use of Carlsbad water the proportion of sugar fell to .6 per cent. Up to this time the gangrene had made considerable progress, but was finally arrested, and replaced by granulations and cicatrices. After an interval of about one month the state of the patient was worse; new gangrenous spots had appeared, always with the same characteristics. The patient then gradually grew worse and died about a month afterward.

Kaposi shows that, as far as gangrene is concerned, it gives origin to the bullæ, and he states that one of the characteristic signs of diabetic gangrene is the appearance, in disseminated spots and in certain regions, of cutaneous lesions of serpiginous course, with consecutive reparation and delimitation to the derma, the above described case being a striking example. At the same time Kaposi insists on the community of origin of all diabetic dermatoses, and claims that their common cause is the impregnation of the tissues with sugar. That the tissues are thus impregnated is beyond question. The excitation caused by this impregnation causes alteration of the sensitive nerves, which gives rise to pruritus; then alteration of the secretory and vaso-motor nerves, as a consequence of which anidrosis, urticaria, etc., are produced, and then, by direct irritation, inflammation of the vascular walls and other tissues. These last present but little tendency to inflammatory proliferation, but, on the contrary, a disposition to necrobiosis. It may be that a feeble, intermittent excretion of sugar, or a diabetes of long duration, may bring about, after a gradual tolerance of the tissues is established, a preponderating proliferation with but feeble destruction of tissue. If this be admitted, Kaposi feels authorized in adding a new variety to the forms of diabetic dermatosis, viz. diabetic papillomatosis. He has seen one case of this in a physician, æt. 60 years, of Rio de Janeiro, who has had diabetes for twenty years.—*Annales de Dermatol. et Syphil.*, Jan. 1884.

The Transmission of Syphilis.

At the close of a paper on this subject, Dr. M. Kassowitz draws the following conclusions:—

1. There are a large number of reported cases, in which women, who have never given any symptoms whatever of syphilis, bring children into the world

which, either at birth or soon after, show signs of infection. This shows the possibility and the reality of the transmission of this disease through the seminal fluid of a syphilitic father to the child.

2. Furthermore, there are a large number of observed cases of women who have borne syphilitic children to syphilitic fathers, and who have since remained uninfected through a number of years; and there are also women among these in whom the paternal transmission has died out. In these few cases the syphilitic contagium has not affected the mother.

3. Clinical observations and experimental inoculations show that such women appear less susceptible to syphilitic infection than other persons who are not placed in such a peculiar connection with a syphilitic organism. But a few cases of infection among such women have been observed.

4. The statements of certain authors as to the syphilitic diseases of mothers, of hereditary syphilitic children, without primary affection, sound as well in relation to the time of the appearance of the first symptoms as they are contrary to nature in the last, and can only be accepted as scientific in a few cases. Further and more exact observations are needed on this subject.

5. There are numerous authentic observations, that women, who are infected with general syphilis of a virulent form during pregnancy, have borne healthy children; and it is thus certain that in these cases the syphilitic poison has not passed the vaginal walls of the mother and the vascular system of the fœtus.

6. Clinical observations teach that these children enjoy a very certain immunity against syphilitic infection. Still there are a few cases of infection of these children during and after birth.

7. Again there are a few observations which show that an infection of the hitherto healthy fœtus (by generation) can take place from the infected mother. *Deutsche med. Wochensch.*, Dec. 19, 1883.

Micro-Organisms from Cases of Purpura Hæmorrhagica.

At the meeting of the Pathological Society of London, of February 19th, Mr. WATSON CHEYNE showed micro-organisms from two cases of purpura hæmorrhagica. One case was that of a girl, æt. 12 years, who had a purpuric eruption with pain in the joints, epistaxis, and bleeding from the ear. The temperature was 104.6° on the morning of death. There were numerous ecchymoses in the skin, and hemorrhage into the pelvis of the right kidney. Sections of the heart showed extensive hemorrhage beneath the exocardium. The capillaries were plugged with small bacilli arranged in colonies; a few were isolated; the typical mode of grouping was evidently in colonies. The walls of the vessels were ruptured; there was no evidence of inflammation. The bacilli measured $\frac{1}{7700}$ in. in length and $\frac{1}{20000}$ in. in diameter. Spores or unstained roundish areas also existed in the rods. There was no peculiarity in the staining reactions. Methylene blue was the best dye. The distension of the wall of the capillaries seemed to show that the bacilli had been growing in the blood for some time. The probable cause of the hemorrhage was the plugging of the vessels with bacilli.

The other case was that of a boy, aged fourteen, who had one brother who bled much at the nose. The patient bled from a boil at first; afterwards there was constant bleeding from the gums, nose, and also into the conjunctiva. The temperature was 98°. After prolonged use of local styptics the bleeding ceased. The anæmia produced was intense, and the patient died. There was hemorrhage from an erosion of one small ulcer in the stomach. There were small subarachnoid hemorrhages. The testes, lungs, brain, skin, serous and mucous membranes were all affected. The lungs, tonsil, and heart were examined.

The pulmonary alveoli were full of blood, and the vessels contained colonies of micro-organisms; the capillaries and some larger vessels were completely blocked with swarms of streptococci, which exhibited a typical chain formation. These micrococci were $\frac{1}{7000}$ in. in diameter. There were no free micrococci. No inflammation and no necrotic layers were seen around the plugged vessels. Sections of the tonsils showed several of the vessels plugged with streptococci. The importance of the observation of two cases of idiopathic purpura hæmorrhagica associated with the presence of micro-organisms was very great. These micro-organisms formed colonies; other kinds did not form vascular plugs (except the typhoid bacillus). The question whether there was an entrance of specific germs from without or whether organisms had penetrated into the blood from the alimentary tract was considered.—*Lancet*, February 23, 1884.

SURGERY.

Antiseptic Surgery in its Applications to Field-Service.

At a meeting of the Woolwich Military Medical Society on February 15, 1884, Surgeon-Major C. H. **Godwin** read a paper on this subject. In the discussion which followed, Sir **Joseph Lister**, who was present, said that it is essential that all the surgeons concerned in the treatment of wounded men should be imbued with the idea of the importance of antiseptic principles. It is of no use for the surgeon who first treats a wounded man to act antiseptically, if the one who follows him does not do so; nor, conversely, is it of any use for the surgeon in the field-hospital to be ever so good an antiseptic surgeon, if his predecessor has defiled the wound. Then, again, as to the other point, in civil practice we may go to any amount of expense almost in our materials; we may have our materials as bulky as we please; but in military practice it is, I conceive, essential that the materials should be cheap, and that they should be, so far as the first treatment of the wounded man is concerned, capable of being employed in small bulk. Reyher divided his cases into the *befingerte* and the *unbefingerte*, cases which had been fingered by somebody before him, and cases which nobody before him had fingered. He said he happened to have had fourteen cases of gunshot-wound of the knee-joint; seven were fingered cases, and seven unfingered. The seven fingered cases had all died except one; he was still living, but he was in high fever, and Dr. Reyher was afraid he might succumb. Of the unfingered cases, all were alive, and only one had had fever, but Dr. Reyher said he had also had a considerable portion of his face shot away; this could not be treated antiseptically, and the fever was so explained, and he said, "Are these not remarkable results?" So much for Reyher and his principle that, as Surgeon-Major Godwin has said, what should be done in the first instance should be simply the covering up of the wound with some antiseptic material, and as little fingering as possible.

I should like to say a word or two with regard to simplifications of the treatment both in the field-hospital and at the front. First, as regards the field-hospital. Allusion has been made to the spray. I have long since held and taught that, of all the parts of the antiseptic system, the spray is the least important. It would not at all break my heart if I were told that I should never be allowed to use the spray again in my life; I am satisfied I could, by other means, get essentially the same results. Many German surgeons do not use the spray; therefore, the fact that the spray is an incumbrance is not, to my mind, an argument against the use of the antiseptic treatment by military surgeons. I